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Visual vertigo syndrome: clinical and posturography findings

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Abstract

Neuro-otological and posturography findings in 15 patients with visually induced vertiginous symptoms (visual vertigo) are reported. Thirteen patients were considered to have a peripheral vestibular disorder; seven had abnormal caloric or rotational test results. Two patients had CNS disorder—a cerebellar degeneration and a brainstem stroke. Posturography testing showed that five patients showed abnormally large body sway induced by full field visual motion stimulation. This group included the two patients with CNS disease and four with strabismic symptoms (diplopia, squint surgery, and ocular muscle weakness). It is concluded that visual vertigo is a heterogeneous syndrome with peripheral or central aetiologies and may occur if patients with balance disorders show high visual field dependence. In patients with visual vertigo, the presence of additional CNS or strabismic symptoms may cause inappropriate postural reactions in environments with conflicting or disorienting visual stimuli, probably by reducing the ability to resolve the sensory conflict.

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The existence of visually mediated vertiginous symptoms is often disregarded because diseases of the eye or visual pathway do not induce any sense of unsteadiness or dizziness. Even in normal subjects, however, certain visual stimuli induce transient spatial disorientation and motion sickness-like symptoms (for example, linear and circular vection, fairground illusions, flight simulators[1]).

Clinicians also know that some patients report "dizzy" symptoms precipitated or exacerbated by certain visual surroundings[2-4] These usually include optic flow stimulation by particularly rich or repetitive visual patterns, such as those encountered in supermarket aisles, unstable visual backgrounds (traffic, crowds, moving objects), and certain driving conditions. Unfortunately, however, many of these patients are discharged after conventional examinations with the impression that a psychological disorder is the underlying cause for their symptoms. In this paper, neuro-otological and posturography findings in 15 such patients are reported.

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Materials and methods

Fifteen patients reporting vertigo, dizziness, or off balance sensations, precipitated or aggravated by certain visual situations, were recruited. Their mean age was 39 (range 21–57) years. All patients underwent a neurological and neuro-otological examination, including eye movement recordings (DC electro-oculography), searching for spontaneous nystagmus, smooth pursuit, optokinetic nystagmus, vestibulo-ocular reflex, and its visual suppression, as described previously.[5] The caloric test was performed according to the technique described by Fitzgerald and Hallpike,[6] with and without Frenzel's glasses, and the duration results were expressed as percentages indicating canal paresis or directional preponderance using standard formulas. Eye movement and caloric results were compared with our normal data.[5]

POSTUROGRAPHY

Body sway in the anteroposterior (x) and lateral (y) axes was assessed by measuring the displacement of the centre of foot pressure. Subjects stood shoeless on an earth fixed force platform with feet 20 cm apart, inside a mobile experimental room mounted on pneumatic wheels. The subject faced one of the lateral walls of the mobile room, at an eye-wall distance of 38 cm, so that displacements of the room provided full field linear optokinetic stimulation along the y, interaural, axis. Discrete room displacements of 30 cm were delivered smoothly by hand with a sigmoid shaped trajectory lasting about one second, peak velocity 2–3 cm/s (peak angular velocity at the level of the eye, 3·76°/s). A potentiometer attached to one of the wheels transduced room displacement.

Baseline body sway recordings, in the absence of room motion, were obtained for one minute with eyes open and closed. Then the room was displaced from right to left, or vice versa, with interstimuli intervals of eight to 16 seconds; four to eight stimuli in each direction were delivered. Body weight was normalised and sway path was measured, defined as the displacement of the combined xy rotating vector of a 70 kg mass per 12 seconds; apparatus and techniques used have been fully described previously.[7 8] Two groups acted as controls for the

posturography protocol described: normal subjects (n = 30, mean age 48 years) and a vestibular control group, without visual vertigo, who underwent unilateral labyrinthectomy or vestibular neurectomy for refractary vertigo six months previously (n = 12, mean age 52 years).

Sway path of the individual patients with visual vertigo were compared with the mean (plus two SDs) of both the normal and labyrinthectomy control groups. The variables assessed were: spontaneous sway path with eyes open (EO), spontaneous sway path with eyes closed (EC), sway path during motion of the room (visual stimulation, VS), and the amount of sway attributed to visual stimulation (VS-EO). Comparisons were also made between sway path values expressed as ratios: "R/L" (ratio between rightwards and leftwards room motion) to identify asymmetries in the visually induced sway response, "2nd/1st" (ratio between the second and first visual stimuli) as a measure of the subject's ability to adapt to the moving visual surrounding, "EC/EO" (Romberg quotient), and "VS/EO" (the ratio between sway during the visual stimuli and sway during eyes open without stimulation) indicative of a subject's sensitivity to the moving surroundings in relation to his or her baseline sway.

Results

CLINICAL AND NEURO-OTOLOGICAL FINDINGS

The more frequent visual precipitating factors or "triggers" were walking in supermarket aisles (six patients), the visual moving surroundings during travelling (five patients in cars and one in trains), moving objects (for example, "disco" lights, people walking, cars passing by; six patients) and movements of the eyes (two patients). It is important to note that most patients reported more than one visual trigger. In nine patients vestibular symptoms not triggered by visual stimuli preceded the appearence of visual vertigo, but in six patients the onset of both types of symptoms was roughly simultaneous. All patients had spontaneous vestibular-like symptoms or unsteadiness, but these were specifically aggravated by the visual conditions mentioned (see case reports later). The duration of the first ranged from six months to 20 years and that of the second from three months to 15 years.

In 10 patients it was thought that the cause of the symptoms was an underlying peripheral vestibular disorder. This diagnosis was reached on a clinical basis in the presence of a reliable history of vertiginous symptoms without evidence of CNS involvement from the history, examination, or imaging procedure. Three further patients were also classified as having possible peripheral vestibular disorder, in the absence of alternative diagnosis at the time of investigations. One of these showed small, scarce lesions in the cerebral white matter on MRI and another patient developed multiple sclerosis two years later. Seven of these 13 patients had caloric/rotational findings beyond our normal limits (a patient with a canal paresis of 7% and six patients with directional preponderances ranging from 14% to 40%) but none of these was a substantial unilateral canal paresis to add sound evidence of a labyrinthine lesion.

Two patients had a CNS disorder at the time of examination, one with clinical and CT evidence of a brainstem stroke and another with a family history and oculomotor disorder indicative of cerebellar degeneration.

POSTUROGRAPHY

Figure 1 shows posturography data. For the purpose of this study, the most meaningful sway variables were VS-EO and VS/EO as these essentially quantify selective unsteadiness in response to visual motion. Ten patients fell within the normal limits of either the normal or labyrinthectomy control groups. Five patients were outside both control limits for those variables (four each for VS-EO and VS/EO, five in all; fig 1).

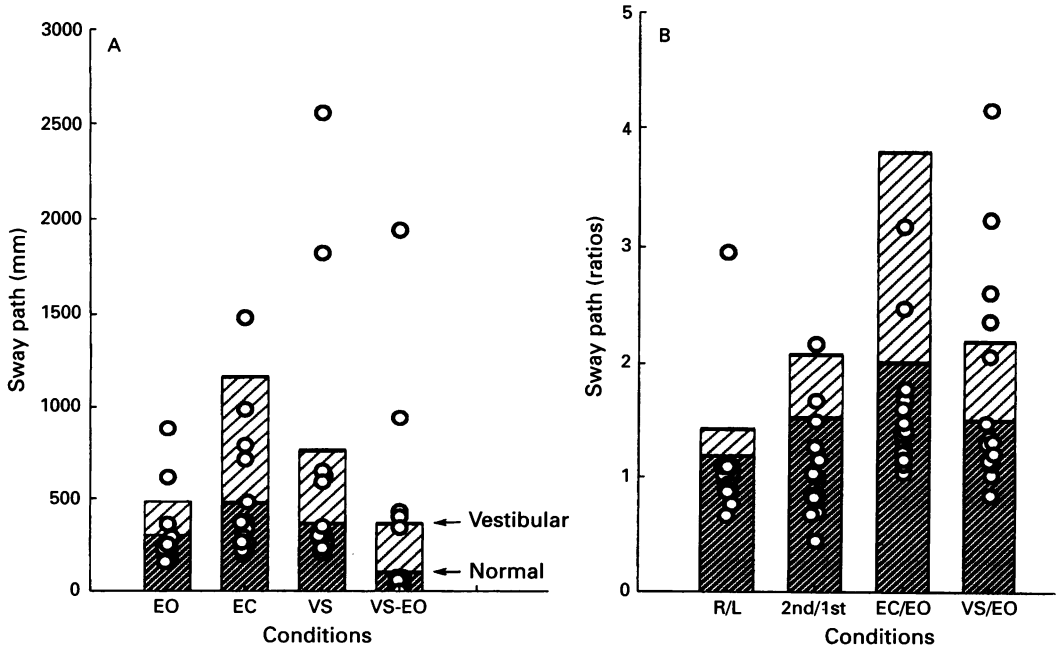

Figure 1 Sway path expressed in mm (left) and as ratios (right) in 15 patients with visual vertigo (open circles). The upper ranges (mean + 2 SDs) in the normal and vestibular control group are indicated by dense and light hatched bars respectively. EO = eyes open; EC= eyes closed; VS = visual stimulus; R/L = ratio between right and left visual stimulus responses; 2nd/1st = ratio between second and first visual stimuli. Four patients with visual vertigo in VS-EO and four in VS/EO (five patients in total) are outside both control groups' limits, indicating enhanced sway responses to the visual motion stimulus.

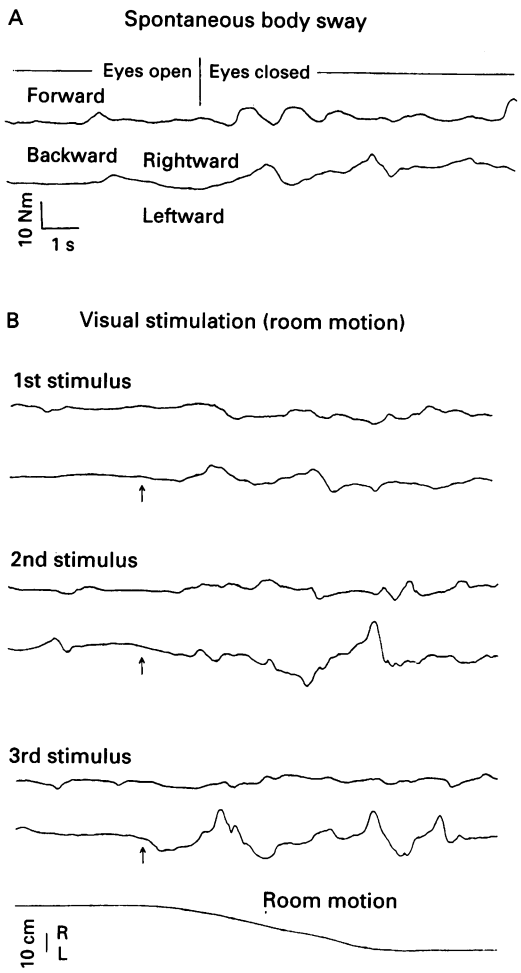

Figure 2 Raw force plate recordings of patient 3. Sagittal (forward-backward) and coronal (rightward-leftward) sway is shown during spontaneous body sway with eyes open and closed (A) and during three consecutive displacements of the mobile room (B). Note the considerable unsteadiness during visual stimulation. The arrows signal stimulus onset.

An attempt was made to identify factors which may explain the selectively increased visually induced unsteadiness in these five patients. Among these patients were the only two with unequivocal cerebellar/brainstem lesions; four of the five patients had either diplopia or extraocular muscle limitations, or both. By contrast, none of the other patients with visual vertigo but normal sway responses had such central or oculomotor findings at the time of examination. Illustrative sway recordings and brief presentations of two cases with exaggerated sway responses follow.

PATIENT NO 3

A 38 year old woman had spontaneous, recurring, short lasting vertiginous episodes and a continuous feeling of being slightly off balance made much worse by visual triggers (fast moving objects, passing cars, modest heights) and head movements for the past six years. Sixteen years earlier she had had a road traffic accident which left her unconscious for a few minutes. She also had had several operations for congenital squint on both eyes, the last one some 10 years earlier. Examination disclosed a slight convergent squint in primary gaze with mild bilateral restriction of abduction, a marginal right sided caloric canal paresis (7%), and a slight high frequency sensory-neural hearing loss on the right. It was thought that the patient's symptoms could be explained by the combination of labyrinthine and extraocular muscle disorders although a clear aetiological diagnosis could not be made.

Figure 2 shows considerable sway induced by room motion in all three consecutive stimuli. Normal responses (reported previously [7 8]) attenuate considerably after the first presentation of the stimulus. Sway path measurements during room movement showed that this patient's values (VS-EO: 431 mm and VS/EO: 3.23) were outside the limits (mean + 2SDs) of both control groups (normal control VS-EO: 100 mm and VS/EO: 1.5; labyrinthectomy control group VS-EO: 360 mm and VS/EO: 2.20).

PATIENT 12

A 54 year old woman had a 15 year history of unsteadiness, which had increased in the past 18 months. Her symptoms were mostly present in moderate heights, supermarkets, and while viewing moving objects. Her mother, sister, and maternal aunt reported balance symptoms but could not be examined. On examination, she had a mild gait ataxia; she reported skew diplopia during examination on gaze left and left-up and a left superior rectus limitation was noted (appearing as a right over left skew deviation). There was gaze evoked nystagmus on looking right, left, and up, abnormal pursuit and vestibulo-ocular reflex suppression, and positioning down-beat nystagmus. Computed tomography and evoked potentials (auditory, somatosensory, and visual) were normal. Magnetic resonance imaging showed multiple non-specific lesions. She was diagnosed as having slowly progressive, familial cerebellar degeneration.

Figure 3 shows raw body sway records of this patient. In this case, prompted by the presence of focusing difficulties which improved on closing one eye during the test, monocular visual stimulation was also investigated. The illustration presents the first stimulus of the series, in this case from left to right, in the sequence in which they were delivered during the same experimental session. The destabilising effect of room motion occurred during binocular and left ("squinting") eye viewing but not during right eye viewing. The effect persisted without habituation during the various stimulus conditions and persisted on two separate testing sessions. Identical effects were seen in this patient during visual stimulation along the fronto-occipital axis (not shown). Sway path measurements during standard, binocular stimulation (VS-EO: 393 mm; S/EO: 2.58) were outside the limits for both control groups.

Discussion

This study was set up to identify neuro-otological or posturographic features which would characterise patients with vertiginous or off balance symptoms triggered by specific visual environments (visual vertigo). The term visual vertigo has also been used to include conditions in which the symptom rather than the trigger is visual (for example, pendular or downbeat nystagmus and head movement

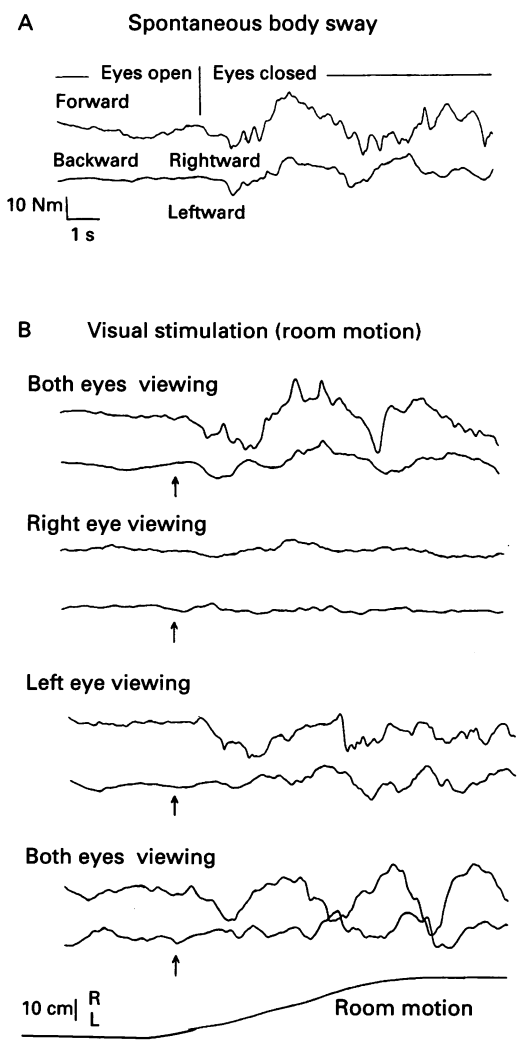

Figure 3 Raw force plate recordings of patient 12 during spontaneous body sway and during visual stimulation. Note the unsteadiness induced by room motion during binocular and left (strabismic) eye viewing but not during right eye viewing. The arrows signal stimulus onset.

induced symptoms in patients with bilateral vestibular loss or oculomotor palsies).[9] In such cases, it may be difficult to distinguish visual vertigo from oscillopsia.

In our patients, labyrinthine, neurological, and strabismic disorders were present, which indicates that visual vertigo is a heterogeneous syndrome. Such heterogeneity warrants further investigation but well established neuro-otological syndromes (for example, positional vertigo) also exhibit varied aetiologies and mechanisms. The question is why some patients with otherwise conventional vestibular disorders exhibit visual "triggers".

THE ORIGIN OF VISUAL VERTIGO

Visually induced postural reactions triggered by external visual motion, as in the experiments described here, are unstabilising with respect to earth-vertical and are normally suppressed by central reweighting of sensory-postural cues.[7 8] Conflicting visual motion stimuli, however, occur in natural (for example, movement of foliage or clouds by the wind) and urban conditions (crowds, traffic), circumstances that bring about off balance feelings in our patients with visual vertigo.

Why patients with visual vertigo are sensitive to particular visual environments is not clear but it is possible that individual idiosyncrasies may play a part. Indeed, tolerance to visual and vestibular stimuli varies greatly even among normal people.[10] Relevant examples are susceptibility to car and sea sickness, fairground rides, and caloric/rotational tests, in which the range of subjective reaction to the same physical stimulus can vary from a pleasant "slight drunkness" feeling to an intolerable life threatening sensation.

An early breakthrough in documenting idiosyncracy in the perception of posturally relevant information came from the work of Asch and Witkin[11] and Witkin.[12] In experiments with normal subjects, in which both the subject and the visual surroundings were independently tilted, it was found that some subjects reoriented themselves to upright driven by the degree of tilt of the visual surroundings whereas others relied more on their own degree of tilt and less on visual cues. The first were called "field dependent", or visually dependent subjects, and the second "field independent".[11 12]

Because idiosyncratic visual and vestibular factors also play a part in the development of motion sickness[10 13] there is every reason to suspect that such normal perceptual differences will also influence the outcome of a lesion causing loss of balance and spatial orientation. A case in point is that of patients with bilateral vestibular loss. In the acute stage these patients' posture is highly sensitive to visual motion but, as compensation gradually develops, a "shift" from a visual to a more proprioceptive mode of postural control takes place[14]—that is, they learn to ignore misleading visual cues by placing more weight on proprioceptive cues for postural control. Visually dependent subjects, however, should have difficulty in shifting from a visual to a proprioceptive mode of operation. Thus it can be postulated that visual vertigo arises when the process of compensation from vestibular lesions is interfered with by high visual dependence, leading to low tolerance to situations of visual conflict. In this context, the finding that a third of our patients showed abnormally large sway responses to visual motion partly supports this view. Future studies must incorporate perceptual assessment, to investigate dissociations between subjective symptoms and sway in these patients, and formal psychological assessment to distinguish visual vertigo from possibly psychogenic disorders such as "postural phobic vertigo".[9]

THE ORIGIN OF ABNORMAL SWAY IN PATIENTS WITH VISUAL VERTIGO

The finding that four of the five patients with visual vertigo with enhanced postural responses to room motion had strabismic symptoms (diplopia, extraocular muscle limitations, strabismus surgery) and that two of the five had brainstem/cerebellar disease may be of relevance. Re-examination of this data in identical experiments in cerebellar and parkinsonian patients[7] showed that CNS involvement itself cannot be the only cause of the increased visually evoked postural responses in the patients with visual vertigo. A CNS disorder associated with strabismic

symptomatology or visual dependence may be required.

Ocular misalignment may play a part in disordered postural control either by an erroneous sense of direction brought about by diplopia or by altered ocular proprioceptive signals.[15 16] During self induced or externally induced optic flow stimulation, the direction of visuopostural responses will be determined by the position of the eye in the orbit and of the head on the trunk. Movement of visual scenes in the frontal plane induces sway in the frontal plane if the subject's eyes and head are in a straight ahead position. If by a combination of eye and head deviation the eyes are, say, at a 90° angle of the trunk, frontal visual motion induces sway along the sagittal plane of the body.[17] This is what is expected if vision is to control posture irrespectively of gaze angle and implies that information from eye and neck proprioceptors have the capability to redistribute visuopostural responses to the required somatic muscles.[18] Thus it could be postulated that ocular misalignment or squint surgery make eye proprioceptive signals unreliable and therefore likely to be disregarded by the postural control centres. Such loss of the regulatory control of eye proprioceptive signals on visuopostural reactions would make these patients further unable to suppress sway responses elicited by conflicting visual stimuli.

It is concluded that patients reporting balance symptoms triggered or exacerbated by certain visual environments require neuro-otological investigation as they are likely to have an underlying abnormality of the vestibular system. It is possible that idiosyncratic features (enhanced visual dependence) make some patients with balance disorders unduly sensitive to visual stimuli. Posturography findings during visual motion partly support this view but further studies incorporating perceptual and psychological assessment are needed.

I am indebted to J Derrick Hood, who introduced me to the problem of visual vertigo and initiated the study of visuopostural reactions when he was Director of the MRC Neuro-otology Unit. Many colleagues at the National Hospital, but especially Gerald Brookes, Linda Luxon, and Peter Rudge, kindly allowed me to investigate their patients.

1 Dichgans J, Brandt Th. Visual-vestibular interaction: effects on self-motion perception and postural control. In: Held R, Leibowitz H, Teuber HL, eds: *Handbook of sensory physiology*. Berlin: Springer-Verlag, 1978: 755–804.
2 Hood JD. Unsteadiness of cerebellar origin: an investigation into the cause. *J Laryngol Otol* 1980;**94**:865–76.
3 Page NGR, Gresty MA. Motorist's vestibular disorientation syndrome. *J Neurol Neurosurg Psychiatry* 1985;**48**: 729–35.
4 Jacob RG, Lilienfeld SO, Furman JMR, Durrant JD, Turner SM. Panic disorder with vestibular dysfunction: further clinical observations and description of space and motion phobic stimuli. *Journal of Anxiety Disorders* 1989; 3:117–30.
5 Lopez L, Bronstein AM, Gresty MA, Rudge P, du Boulay EPGH. Torsional nystagmus. A neuro-otological and MRI study of thirty-five cases. *Brain* 1992;**115**:1107–24.
6 Fitzgerald G, Hallpike CS. Observations on the directional preponderance of caloric nystagmus resulting from cerebral lesions. *Brain* 1942;**65**:115–37.
7 Bronstein AM, Hood JD, Gresty MA, Panagi C. Visual control of balance in cerebellar and Parkinsonian syndromes. *Brain* 1990;**113**:767–79.
8 Bronstein AM. Suppression of visually evoked postural responses. *Experimental Brain Research* 1986;**63**:655–8.
9 Brandt Th. *Vertigo*. Berlin: Springer-Verlag,1991.
10 Benson AJ. Motion sickness. In: Dix MR, Hood JD, eds. *Vertigo*. New York: John Wiley, 1984:391–426.
11 Asch SE, Witkin HA. Studies in space orientation. II. Perception of the upright with displaced visual fields and with body tilted. *Journal of Experimental Psychology* 1948; **38**:455–77.
12 Witkin HA. The perception of the upright. *Sci Am* 1959; **200**:51–6.
13 Yardley L. Motion sickness susceptibility and the utilisation of visual and otolithic information for orientation. *Eur Arch Otorhinolaryngol* 1990;**247**:300–4.
14 Bles W, De Jong JMBV, de Wit G. Compensation for labyrinthine defects examined by use of a tilting room. *Acta Otolaryngol (Stockh)* 1983;**95**:576–9.
15 Gauthier GM, Nommay D, Vercher J-L. The role of ocular muscle proprioception in visual localization of targets. *Science* 1990;**249**:58–60.
16 Steinbach MJ, Smith DR. Spatial localization after strabismus surgery: evidence for inflow. *Science* 1981;**213**: 1407-9.
17 Bronstein AM. Visually induced body sway in patients with visual vertigo. In: Taguchi K, Igarashi M, Mori S, eds. *Vestibular and neural front, proceedings of the 12th International symposium on posture and gait, Matsumoto, Japan, 1994*. Amsterdam: Elsevier, 1994:569-72.
18 Roll JP, Roll R. From eye to foot: a proprioceptive chain involved in postural control. In: Amlard B, Berthoz A, Clarac F, eds. *Posture and gait: development, adaptation and modulation*. Netherlands: Elsevier Science, 1988: 55–164.